Amendments to the Specification:

Delete the title at page 1, line 1 and replace it with the following new title:

METHODS OF MODULATING T CELL OR NATURAL KILLER CELL ACTIVITY WITH ANTI-P-SELECTIN GLYCOPROTEIN LIGAND 1 ANTIBODIES

Replace the paragraph beginning at page 7, line 1 with the following amended paragraph:

Fig. 5 depicts the levels of IL-2 produced in mixed lymphocyte culture using spleen cells isolated from TAB4 (or hamster Ig)-treated BALB/c ~~Balb/c~~ mice as the responders and H2-mismatched C3H spleen cells as the stimulator.

Replace the paragraph beginning at page 7, line 8 with the following amended paragraph:

Fig. 7 depicts the percentage of surviving grafts in C57BL/6 mice that received a skin graft from BALB/c ~~Balb/c~~ mice and were treated with an anti-PSGL-1 antibody (closed diamond) or a control antibody (open square).

Replace the paragraph beginning at page 17, line 17 with the following amended paragraph:

A TAIP-specific monoclonal antibody was generated by applying the well known cell fusion methods of Kohler and Milstein ((1976) European Journal of Immunology 6:511-519) to produce a hybridoma secreting desired antibodies. Antibody-producing cells from a hamster injected with Concanovalin A (Con A)-activated BALB/c ~~Balb/c~~ spleen T cells were fused with a myeloma cell line to form an antibody secreting hybridoma. The two populations of cells were fused with polyethylene glycol, and the resulting antibody producing cells were cloned and propagated by standard tissue culture methods. One hybridoma generated according to these methods secreted a monoclonal antibody, designated TAB4, that was able to induce T cell apoptosis *in vitro* and deplete T cells *in vivo*. The protein recognized by TAB4 was designated T cell apoptosis inducing protein (TAIP).

Replace the paragraph beginning at page 24, line 8 with the following amended paragraph:

BALB/c ~~Balb/c~~ mice (H-2d) were intraperitoneally injected with 300 micrograms of TAB4 or control hamster Ig. Splenocytes were isolated 7 days after injection, and used as responders in culture with mitomycin C-treated C3H (H-2k) splenocytes (as stimulators). Three days later, the culture supernatants were harvested and the IL-2 content was measured by ELISA set (PharMingen). As shown in Fig. 5, the IL-2 production was suppressed in responder cells derived from TAB4-treated mice as compared with that of control mice. The plasma levels of IL-2 and TNF-a were also analyzed and no significant difference was noted in the levels of IL-2 (or TNF-a) in the sera of the control and the TAB4 treated mice. Since production of IL-2 is central to the activity of T cells, the results show that a TAIP-specific antibody, such as TAB4, can be used *in vivo* to manipulate T cells and control unwanted T cell-mediated immune responses such as those associated with autoimmune diseases and transplantation rejection.

Replace the paragraph beginning at page 25, line 5 with the following amended paragraph:

Mice (obtained from Jackson Laboratory) at 8 to 12 weeks of age were anesthetized with Acepromazin maleate (Fermenta Animal Health Co., Kansas City, MO). Prior to skin grafting, non-thymectomized recipient C57BL/6 mice ($H\text{-}2^b$) were injected intraperitoneally with 500 ug of TAB4 or isotype control antibodies seven days before skin transplant surgery. Seven days later, a lateral flank of skin from fully allogeneic mismatched BALB/cj ~~Balb/cj~~ mice ($H\text{-}2^d$) was grafted on the lateral flank of the antibody pre-treated C57BL/6 mice. Seven days post transplantation, the mice were again injected with 500 ug of TAB4 or isotype control antibody. The mice were monitored every day after graft transplantation. The grafts were considered rejected when 50% donor skin was necrotic. The percent of graft survival is shown in Fig. 7 (n=8). The data show that TAB4 antibody treatments prolonged the survival of the allogeneic skin grafts.